

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	32	luo-ying.in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/07/08 14:19
L3	0	"xu-xiang.in"	US-PGPUB; USPAT	OR	ON	2005/07/08 14:24
L5	57	TRAF4	US-PGPUB; USPAT	OR	ON	2005/07/08 14:26
L6	765	TRAF	US-PGPUB; USPAT	OR	ON	2005/07/08 14:26
L7	2	Mkinase	US-PGPUB; USPAT	OR	ON	2005/07/08 14:31
L8	3	Mkinase	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/07/08 14:32

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 14:39:49 ON 08 JUL 2005

L1 245 S LUO YING/AU

L2 178 S XU XIANG/AU

L3 1 S MKINASE

L4 4 S TRAF4(S) INTERACTING (S) PROTEIN?

L5 182 DUP REM L1 (63 DUPLICATES REMOVED)

L6 146 DUP REM L2 (32 DUPLICATES REMOVED)

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COST IN U.S. DOLLARS

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0.21	0.21

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L1 245 LUO YING/AU

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=> dup rem L1
PROCESSING COMPLETED FOR L1
L5 182 DUP REM L1 (63 DUPLICATES REMOVED)

=> dup rem L2
PROCESSING COMPLETED FOR L2
L6 146 DUP REM L2 (32 DUPLICATES REMOVED)

=> d ibib abs L3

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:229044 CAPLUS
DOCUMENT NUMBER: 134:265156
TITLE: Traf4 associated cell cycle proteins, compositions and
methods of use
INVENTOR(S): Luo, Ying; Huang, Betty
PATENT ASSIGNEE(S): Rigel Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001021799	A1	20010329	WO 2000-US40987	20000925
WO 2001021799	C2	20020808		
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2385879	AA	20010329	CA 2000-2385879	20000925
EP 1218506	A1	20020703	EP 2000-975649	20000925
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, FI, CY
JP 2003510049 T2 20030318 JP 2001-525357 20000925
PRIORITY APPLN. INFO.: US 1999-404010 A 19990923
WO 2000-US40987 W 20000925

AB The present invention is directed to novel polypeptides, nucleic acids and related mols. which have an effect on or are related to the cell cycle. The novel cell cycle protein is a **Mkinase** and binds to Traf4, a tumor necrosis factor receptor-associated factor. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide mols. comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention. Further provided by the present invention are methods for identifying novel compns. which mediate cell cycle bioactivity, and the use of such compns. in diagnosis and treatment of disease, e.g. induce apoptosis of tumor and cell proliferation in wound healing.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs L4

L4 ANSWER 1 OF 4 MEDLINE on STN
ACCESSION NUMBER: 2002396330 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12023963
TITLE: Involvement of TRAF4 in oxidative activation of c-Jun N-terminal kinase.
AUTHOR: Xu You Cheng; Wu Ru Feng; Gu Ying; Yang Yih-Sheng; Yang Meng-Chun; Nwariaku Fiemu E; Terada Lance S
CORPORATE SOURCE: Department of Internal Medicine, University of Texas Southwestern and The Dallas Veterans Affairs Medical Center, Dallas, Texas 75216, USA.
CONTRACT NUMBER: R01-HL61897 (NHLBI)
SOURCE: Journal of biological chemistry, (2002 Aug 2) 277 (31) 28051-7. Electronic Publication: 2002-05-22. Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020730
Last Updated on STN: 20030105
Entered Medline: 20020916

AB We previously found that the angiogenic factors TNFalpha and HIV-1 Tat activate an NAD(P)H oxidase in endothelial cells, which operates upstream of c-Jun N-terminal kinase (JNK), a MAPK involved in the determination of cell fate. To further understand oxidant-related signaling pathways, we screened lung and endothelial cell libraries for interaction partners of p47(phox) and recovered the orphan adapter TNF receptor-associated factor 4 (TRAF4). Domain analysis suggested a tail-to-tail interaction between the C terminus of p47(phox) and the conserved TRAF domain of TRAF4. In addition, TRAF4, like p47(phox), was recovered largely in the cytoskeleton/membrane fraction. Coexpression of p47(phox) and TRAF4 increased oxidant production and JNK activation, whereas each alone had minimal effect. In addition, a fusion between p47(phox) and the TRAF4 C terminus constitutively activated JNK, and this activation was decreased by the antioxidant N-acetyl cysteine. In contrast, overexpression of the p47(phox) binding domain of TRAF4 blocked endothelial cell JNK activation by TNFalpha and HIV-1 Tat, suggesting an uncoupling of p47(phox) from upstream signaling events. A secondary screen of endothelial cell **proteins for TRAF4-interacting partners** yielded a number of **proteins** known to control cell fate. We

conclude that endothelial cell agonists such as TNFalpha and HIV-1 Tat initiate signals that enter basic signaling cassettes at the level of TRAF4 and an NAD(P)H oxidase. We speculate that endothelial cells may target endogenous oxidant production to specific sites critical to cytokine signaling as a mechanism for increasing signal specificity and decreasing toxicity of these reactive species.

=> d his

(FILE 'HOME' ENTERED AT 14:39:20 ON 08 JUL 2005)

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